AD NUMBER

AD836227

NEW LIMITATION CHANGE

TO

Approved for public release, distribution unlimited

FROM

Distribution authorized to U.S. Gov't. agencies and their contractors; Foreign Government Information; NOV 1963. Other requests shall be referred to Department of the Army, Fort Detrick, MD 21701.

AUTHORITY

SMUFD D/A ltr, 8 Feb 1972

TRANSLATION NO. F96

DATE: Nov 1965

DEPARTMENT OF THE ARMY
Fort Detrick
Frederick, Maryland

ANTHRAX EXPERIMENTS

/Following is the translation of an article by Prof. Dr. Kurt Schern, Montevideo, in the German-language periodical Berliner Tieraerztliche Wochenschrift (Berlin Veterinary Weekly), Berlin, August 1936, pages 569-570./

The experiments on which I would like to report in brief below were conducted between 1925 and 1928. For the moment, however, I am not in a position to continue my anthrax experiments due to other pressing business. I would therefore like to present at least a brief report on results achieved to date in these experiments.

As we know, the brilliant, still unsurpassed method employed by Pasteur in the production of a vaccine against anthrax is based on the reduction of the virulence of the cultures through prolonged breeding at certain temperatures. Recently, Bezredka recommended the cutaneous use of the vaccine on whose results in Germany we have a report by Bartels (Deutsche Tieraerztliche Wochenschrift /German Veterinary Weekly/.)

(Bartels' report, by the way, corrects a misapprehension which has been widespread since the war. Nature itself has disproved the contrived allegation that Germany's blockade from 1914 to 1918 was responsible for the decrease in anthrax cases in certain regions. It was asserted at the time that the absence of imports of foreign and thus partly anthrax-infected hides caused the drop in anthrax cases. Instead, it turned out that -- although no hides were imported from abroad between 1931 and 1933 -- there was an increase in anthrax cases again in 1934 in the particular regions involved. In 1934, 22 cases of anthrax were observed in cattle in these regions and in 1933 the number was 13. This is why people believed that the observations made in connection with anthrax were due to the reduced imports of hides during and after the war; of course, this was wrong. In my opinion, we cannot say today that we did not get any or only isolated anthrax reports simply because all our veterinarians had been drafted into the army and were on duty in the field. The same mistaken interpretation was made during the war in connection with the reduced incidence of hoof-and-mouth disease.)

Schern, a student of Uhlenhut, has laid the foundation for an entirely new theory on the effect of trypanosoma, etc., in warm-blooded organisms; Schern worked on this project in Uhlenhut's laboratory. Today the theory conceived at that time has been expanded and proved experimentally to a point where we can say the following: the parasites split the carbohydrates in the organism; they alter the carbohydrate metabolism so thoroughly that the entire liver balance breaks down in time and leads to death. These facts have caused Schern and Artagaveytia to subject many substances producing hypoglycemic effects to systematic study over a number of years; they conducted experiments in an effort to develop a glycoprival therapy for the infections involved. As a result, syntalin was found to be remedy against these diseases; this was discovered by N. and H. von Jancso, using Schern's theory on the disturbance in sugar metabolism, in the course of infections observed in Hungary -- although the authors did not know of each other's work. The two first-named authors also used syntalin as remedy in the case of Spironema hispanicum (Sociedad de Biologia de Montevideo, 1935).

In the case of anthrax, our work was bound to run along different lines. It was to be assumed that the anthrax bacillus operates differently in the infected organism than does trypanosoma, etc. The very finding to be made in anthrax cadavers points to this. This further leads to the idea that anthrax involves a fermentative cleavage of albumin particles.

If we put Pasteur cultures in milk, expose them to incubation at 37° C, and if, for comparison, we put highly virulent anthrax cultures in milk under the same conditions and during the same span of time, we find a basic difference between these two culture groups; there are some reports on this in the literature on the subject. The Pasteur vaccine strains digest milk very slowly. On the other hand, the highly virulent culture, bred freshly from large animals that died of anthrax (cattle, sheep, etc.), digests milk in a remarkably short time so that one is tempted to draw a parallel between virulence and digestive capacity. We can see something similar in the peptonization of gelatins through anthrax, if we inoculate them with various virulent strains and if we incubate them. However, we can observe this albumin decomposition much better in pure casein solutions. Ground heart muscle flesh and muscle flesh in general, etc., is digested by highly virulent anthrax bacteria much faster than by little virulent bacilli of this kind.

Anyone whose job it is to determine the virulence of anthrax bacilli in practice can save considerable expense by first determining the digestive capacity of the particular strain with respect to casein and by establishing a proportion between the casein-digestion capacity and the animal-killing capacity according to a scheme worked out in advance in suitable experiments. In regions where we have much inoculation against anthrax, we will have to determine whether an animal has died of vaccine anthrax or virulent anthrax. This question must also be answered in the case of strains isolated from skins. The casein-digestion capacity here offers us valuable and inexpensive indications.

As we said before, the fermentative albumin decomposition in the culture due to the anthrax bacilli corresponds, as to its speed, to the particular virulence; this of course leads us to the following questions: What is the effect of the anthrax bacilli in the anthrax-infected warm-blooded organism? Do its enzymes also attack the albumin of the organism, as in the culture? To tackle these questions, I infected a rabbit with anthrax and tested the blood, respectively, the serum, for the amount of precipitable albumin and the amount of peptone contained in these substances in comparison to normal rabbit blood, etc. I found that the serum of normal animals contains twice the quantity of precipitable albumin than is usually obtained from the serum of infected animals. The tremendous change in the composition of the serum explains why it can no longer perform its function in the diseased organism and why death occurs. The peptone experiments did not yield definite results; nevertheless, there is some justification -- within certain limits -- in concluding that the peptones and the serum of the anthrax-infected animal are considerably increased.

I then went one step further and tried to determine the effect of the anthrax antiserum. I allowed this antiserum to act for a certain time (5 hours) on 1 cc of a fresh suspension of highly virulent anthrax bacilli. Then I centrifuged the bacteria and tested them as to their digestive capacity in comparison to bacteria from the same source which, however, had been kept in the incubation cabinet with the normal serum of the same animal species, under the same conditions and for the same period of time. I tested the digestive capacity on casein solutions, using the same bacteria and casein quantities. I found that the highly virulent anthrax bacteria, which had been treated with anthrax antiserum, leave twice to three times the quantity of casein undigested -- in comparison to the quantity digested by bacteria treated with normal serum. Thus the digestive capacity of bacteria treated with antiserum is inhibited twice to three times. We can prove the digestion-inhibiting effect of the antiserum visavis virulent bacteria also in gelatin experiments but the phenomena here are not as obvious as in the case of casein. This means that we have bodies in the anthrax antiserum which act against the digestion of the anthrax bacillus. (I ought to point out that I did not have all of our modern literature available.) Using the methods described above we can easily determine the digestion-inhibiting capacity of anthrax antiserum. But, since digestive capacity and virulence are presumably related, there is a great temptation here to arrive at certain conclusions. These will seem justified only the moment my findings have been generally confirmed and as soon as certain other points have been cleared up. The procedure, based on the above method, will probably be the same for the other "anti-infection sera."

If we inject rabbits subcutaneously with precisely dosed trypsin quantities, the animals die and reveal similar phenomena as animals infected with anthrax. We can note an edema, similar to anthrax, at the

point of injection; it is however not as gelatinous as in an animal killed with 1/1,000,000 loop of anthrax. If we realize the difficulties involved in the production of each enzyme-antiserum, we will -- if we consider the enzyme character of the anthrax bacillus -- also understand why the preparation of a good anthrax antiserum is not easy. The obstacles here are created by the anthrax bacillus with its particular character as an infectious digestion enzyme. In standardizing the virulence of the anthrax bacteria and of anthrax antiserum we can also use trypsin.